

Use of MS-H Vaccine in

Broiler Breeders

Peter C. Scott, BSc, BVSc, PhD, School of Veterinary Science, University of Melbourne, Australia

Mycoplasma synoviae (MS) is usually considered by poultry producers as a clinically inapparent respiratory infection of poultry. Many, though, have experienced severe economic losses due to MS and have come to recognise it as a significant pathogen within their operations.

Part of this realisation has been associated with better diagnostic capabilities, and also with the absence of complicating or masking disease agents like Mycoplasma gallisepticum (MG), Infectious Coryza, and Fowl Cholera. In fact, it has become clear that the presence of MS can result in a greater expression of clinical disease associated with these other primary avian pathogens.

The attitude of producers towards MS as a cause of disease is determined by the nature of the isolate within their organisation, and this can vary

considerably. The presence of MS within commercial poultry can vary from no clinical disease, a mild respiratory snick in the cool and increasing humidity of the evening in breeders or layers, to a severe suppurative synovitis in parent breeder rearers or pullets.

In broiler breeders challenged with MS throughout rearing or in lay, the subsequent vertical shedding to broiler progeny can result in serious economic losses with morbidity in some flocks being up to 70%.

Unfortunately, this shedding can continue intermittently and at various rates throughout the lifetime of the donor flock as it undergoes other production and husbandry stresses.

Depending on the strain or isolate of MS, clinical disease can manifest as a severe suppurative synovitis affecting a variety of joints, classically the hock joint, or it can manifest in a more insidious form

complicated with colibacillosis associated CRD.

In this latter form the high prevalence of a pericarditis without the normally associated severe perihepatitis/airsacculitis should alert the producer to a possible involvement of MS.

It was the success of the chemically attenuated live MG vaccine strain ts-11 that led to the University of Melbourne's applying similar methodology to the production of an attenuated MS vaccine. A field isolate of MS was subjected to chemical mutagenesis with nitrosoguanidine and the mutants selected by looking for temperature-sensitive phenotypes (ts+).

A clone that was stable through in vitro and in vivo passaging, and demonstrated no reversion to virulence or loss of temperature phenotype, was chosen as a vaccine candidate for further evaluation.

Subsequent safety and efficacy testing within the laboratory, followed by field evaluation, resulted in the commercially available MS-H vaccine. Readers can review the development of the MS-H vaccine in the following papers (2, 3, 4, 5, 6, 9).

In Australia in the 1980s, most broiler parents were positive for MG necessitating the extensive use of tylosin in both parents and their progeny to try to sustain a reasonable level of productivity. The introduction of improved husbandry and biosecurity practises in conjunction with the use of the attenuated live MG vaccine ts-11 has now meant that essentially all parent broiler breeders and parent layers are negative for MG in Australia, and the use of tylosin for the treatment of clinical mycoplasmosis is a rarity.

Similarly, the introduction of MS-H into the broiler breeder parent population has meant that clinical disease related to MS is essentially not recognised.

The implementation of MS-H vaccination in commercial layers has been less and is in part due to the difficulties in measuring the performance impacts of MS infection in commercial flocks because of the lower virulence of MS strains involved and the maintenance of birds under optimal housing conditions.

With the emergence of more extensive alternative layer systems, this scenario is already beginning to change.

In discussing eradication or control programs for MS in parent stock, two situations may exist in commercial poultry flocks. First, progeny is being obtained from donor flocks that are positive for mycoplasma.

Once birds are positive for mycoplasma, vaccination with attenuated live vaccines is of no value because it needs to colonise them prior to challenge with a wild-type mycoplasma. Positive donor flocks should be preferably utilised for progeny production during mid-lay and not during periods of production stress like early lay.

The donor flock is medicated with an antibiotic to which the mycoplasma is susceptible for 7 days prior to the collection of hatching eggs. The dosage level should be mycoplasmacidal in the egg, and medication continued for the duration of the hatching egg accumulation period.

Tiamulin at 32 mg/kg live bird weight/day is considered mycoplasmacidal in the egg. Day-old chickens, are then delivered to the brooding facility which has been cleaned and sanitised and maintained biosecure.



Where a single age farm cannot be achieved, while the risk assessment is higher, movement towards mycoplasma freedom with this program can be still be achieved. Progeny should then be treated for 3 consecutive days with soluble lincomycin-spectinomycin and then treated again from day 7 to 10 with, for example, tylosin. Around 7 days post-withdrawal of medication all birds are then vaccinated with MS-H (and/ or ts-11) according to the recommended instructions for vaccine handling and application.

After this, sound biosecurity practices should be maintained. No medications to which mycoplasma are susceptible should ever be used in the flock while attempting this movement toward clearing wild-type mycoplasma from the operation. It should be emphasised at this point that the above protocol is not intended to be scientifically rigid or text book in its descriptive process, but is aimed at allowing some progression toward freedom from mycoplasma disease in a low cost manner under commercial conditions that do not, in the short-term, allow obtainment of optimal status livestock that are placed in optimal facilities under preferred husbandry situations.

The second, more regular situation is where progeny is obtained from negative

donors, but this status is lost either in rear or during production. While work at the University of Melbourne indicates that it is preferable to vaccinate birds from around 6 to 12 weeks of age with the live attenuated mycoplasma vaccines to achieve optimal vaccine efficacy, this slight laboratory-measured advantage should be considered only with reference to on-farm situations.

With negative progeny on very biosecure farms, vaccination can be done later, but certainly a minimum of 4 weeks before the birds are put at risk of any wild-type exposure. On facilities or in operations where biosecurity breaches are common and seroconversion is noted earlier in rear, vaccination should be considered as soon after 14 days as possible. In all circumstances, the use of antibiotics to which mycoplasma are susceptible is contraindicated.

Producers, either by implementing an eradication program or obtaining and maintaining negative mycoplasma stock, should not make the common mistake of stopping vaccination because they now do not have a problem. This is, unfortunately, a relatively common occurrence where, after elimination of mycoplasma disease, the producer ceases vaccination, the principle component in his control

program. This is even more concerning when the deficient aspects of biosecurity and husbandry have not been addressed.

Other advantages of MS-H is that it demonstrates very minimal horizontal spread and then only under conditions of very close contact; and also no evidence of vertical transmission has been detected in pipped hatching eggs or progeny from MS-H vaccinated parents.

For this reason, operators who need to achieve a status of mycoplasma freedom in, for example, elite breeding stock can achieve this by using MS-H to displace the wild-type organism.

Once confidence is gained about wild-type status, husbandry, and biosecurity practises, they can withdraw vaccination. Cessation of vaccination should not be considered if facilities and biosecurity practises indicate a high-risk assessment.

The efficacy of this vaccine has been demonstrated to be dose dependent (3); therefore, any loss of vaccine titre through inappropriate handling techniques or sub dose administration will reduce its

protective index. It is critical that veterinarians and vaccination crew supervisors ensure, as is done with Marek's disease vaccination protocols, that no sub-standard procedures are in place when live attenuated mycoplasma vaccines are being used.

It is known that for these live attenuated mycoplasma vaccines to be efficacious they need to successfully colonise in the upper respiratory tract and establish their host relationship before any challenge from wild-type mycoplasma.

Avian mycoplasmas are able to colonise in their host indefinitely because they have developed mechanisms to escape immune recognition by the host.

This ability to change antigenic surface proteins is controlled by a variable gene region. It is for this reason that one successful vaccination with MS-H is all that is required for life long protection of commercial fowl.

In fact, it may be contraindicated to attempt to revaccinate a bird once it has been colonised with an attenuated live mycoplasma vaccine.

The immune mechanisms operating in avian mycoplasma infections are not entirely understood.

While immunity to MS is bursal-dependent, protection is probably not due to antibodies circulating in the blood. Respiratory secretory antibodies and/or cell mediated mechanisms may play a role (1, 7).

Compared to killed mycoplasma vaccines, the serological response to vaccination with MS-H using the Rapid Slide Agglutination (RSA) test results in low to variable results. This is considered normal and expected as the protective immune response mediated by MS-H results from its non-invasive colonisation of the upper respiratory tract as an attenuated mycoplasma vaccine.

Seroconversion after MS-H vaccination is also usually noted to be slow, taking up to six weeks before a typical post-vaccination response is seen.

More details on interpretation of serological findings after vaccination with the live attenuated vaccines ts-11 and MS-H can be reviewed in literature (8).

References

1. Kume, K., Y. Kawakubo, C. Morita, E. Hayatsu and M. Yoshioka. Experimentally Induced Synovitis of Chickens with *Mycoplasma synoviae*: Effects of Bursectomy and Thymectomy on Course of Infection for the First Four Weeks. *Am. J. Vet. Res.* **38**:1595-1599, 1977.
2. Markham, J. F., C. J. Morrow, P. C. Scott and K. G. Whithear. Safety of a Temperature-sensitive Clone of *Mycoplasma synoviae* as a Live Vaccine. *Avian Dis.* **42**:677-681, 1998.
3. Markham, J. F., C. J. Morrow and K. G. Whithear. Efficacy of a Temperature-sensitive *Mycoplasma synoviae* Live Vaccine. *Avian Dis.* **42**:671-767, 1998.
4. Markham, J. F., P. C. Scott and K. G. Whithear. Field Evaluation of the Safety and Efficacy of a temperature-sensitive *Mycoplasma synoviae* Live Vaccine. *Avian Dis.* **42**:682-689, 1998.
5. Morrow, C. J., Pathogenicity, immunity and strain identification of Australian isolates of *Mycoplasma synoviae*. PhD Thesis, University of Melbourne, 1990.
6. Morrow C. J., J. F. Markham and K. G. Whithear. Production of Temperature-sensitive Clones of *Mycoplasma synoviae* for Evaluation as Live Vaccines. *Avian Dis.* **42**:667-670, 1998.
7. Scott, P. C. Desarrollo De Mutante Termiosensible de *Mycoplasma synoviae*. (MS-H). Y su aplicacion en el Campo Como Vacuna Viva Atenuada. Proceedings of VIII Curso de Actualizacion Avi - Mex, Mexico City, Mexico. 1996.
8. Scott, P. C., J. F. Markham, A. H. Noormohammadi and K. G. Whithear. Serological Response to Laboratory and Field Vaccination of Chickens with Two Live *Mycoplasma* Vaccines ts-11 (*Mycoplasma gallisepticum*) and MS-H (*Mycoplasma synoviae*). *Proc. West. Poult. Dis. Conf.* **48**:77, 1999.
9. Whithear, K. G. Control of avian mycoplasmoses by vaccination. *Rev. sci. tech. Off. Int. Epiz.*, **15**(4):1527-1553, 1996.